Hudson (E. D.)

THE

PATHOLOGY AND ETIOLOGY

OF

PULMONARY PHTHISIS,

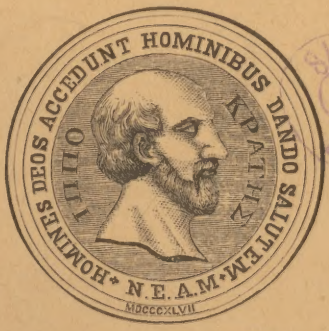
IN RELATION TO ITS PREVENTION AND EARLY ARREST.

BY

E. DARWIN HUDSON, JR., A. B., M. D.,

PROFESSOR OF THE PRINCIPLES AND PRACTICE OF MEDICINE, WOMAN'S MEDICAL COLLEGE OF THE NEW YORK INFIRMARY.

[*REPRINTED FROM THE TRANSACTIONS OF THE NEW YORK ACADEMY OF MEDICINE.*]



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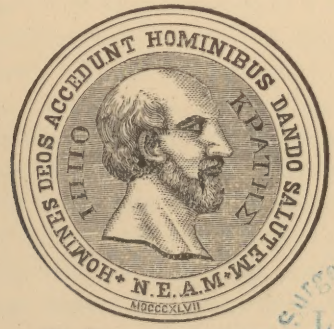

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Read April 1, 1875.

The maladies which have always been the chief enemies of man are the contagious diseases, continued fevers, and pulmonary phthisis. In all ages they have been subjects of constant observation and thoughtful study.

But following Bayle,[1] Laennec,[2] and Louis,[3] a general belief in the specific nature of phthisis seems, at least for a time, to have ended controversy as to its etiology, and to have discouraged the question of its prevention and early arrest.

Bayle and Laennec were victims of the disease they had scientifically defined. Public records and individual observation testify to its preëminence as a destroyer of human life. "It is the chronic plague of the prime of life."[4] In many of the older and populous communities of this country, the mortality from phthisis is one-fifth of deaths from all causes.[5]

Yet there are facts which warrant the belief that phthisis has definite predisposing and exciting causes. To ascertain and diminish these causes is a duty in this age of sanitary science.

[1] "Recherche sur la Phthisie Pulmonaire," 1810.
[2] "Traité de l'Auscultation Médiate," etc., 1819.
[3] "Recherches Anatomico-pathologiques sur la Phthisie," 1825.
[4] "Lectures on Public Health," Dr. Guy, London, 1870.
[5] United States Census, 1870, Washington, 1872.

Pathology.—The history of phthisis is that of tubercle with associated or secondary lesions.

The gross appearances of yellow tubercle have been known for centuries.

Gray or miliary tubercle was first described by Stark,[1] in 1785, by Baillie,[2] in 1794, and more fully by Bayle,[3] in 1810. Tubercle, in these two forms, and the changes through which it passed, has been considered the pathology of pulmonary phthisis. Laennec, in 1819, considered it always a specific disease. Louis accepted the pathology of Bayle and Laennec, and devoted seven years to close observation of the clinical history of the disease. He may be said to be the great advocate of the tubercular nature and unity of phthisis. His views are quite generally held, in this country, at the present day, though elsewhere eradicated by the steady advances of pathology. But independent investigators in France, and especially in Germany, while recognizing the contributions of Laennec and Louis to the diagnosis and symptomatology of phthisis, doubted their views on pathology. Andral,[4] in 1836, said phthisis might occur in persons not predisposed, and in good health, from simple inflammatory processes. Carswell,[5] in 1838, said gray need not precede opaque tubercle. Addison,[6] in 1845, described crude tubercle as a degenerated inflammatory product. The analysis by Preuss [7] proved yellow tubercle to be rich in caseine. Sherer and Lehmann found in different specimens variable quantities of fat. The study of inflammation discovered that fatty metamorphosis is the fate of unabsorbed, unorganized, exudation matter. The charac-

[1] "Handbuch der Allgemeinen Pathologie," Uhle and Wagner, p. 479, 1872.

[2] "Morbid Anatomy," Matthew Baillie, London, 1794.

[3] *Op. cit.*

[4] "Cours de Pathologie Interne," Paris, 1836.

[5] Sir Robert Carswell, "Illustrations of Elementary Forms of Disease," 1838.

[6] "Transactions of Provincial Medical and Surgical Association," 1845.

[7] "Chemical Analysis of Tubercle," by Preuss, Peaslee's "Histology," 1858.

teristics of crude tubercle under the microscope and by chemical analysis were those of metamorphosed inflammatory products. Reinhart,[1] in 1850, advocated their identity. Lebert,[2] in 1851, termed crude tubercle inspissated pus.

Hence the conclusion that "crude tubercle" may occur without the intervention of gray tubercle, a metamorphosis of catarrhal and pneumonic products,[3] and a classification of pulmonary phthisis into—

1. Tubercular
2. Simple inflammatory

} forms.

Still, the indoctrinated faith in the specific nature of phthisis held its ground and endeavored by various theories to connect caseous products in the lung with true tubercular dyscrasia. The *post-mortem* frequence of caseous masses and infrequence of gray tubercle could not be denied. But gray tubercle was defined to be "nascent," "germinal," transitory in office and duration, tending to retrograde at the very beginning of its existence. On the periphery of caseous masses, and in their immediate vicinity, miliary tubercles, some undergoing degeneration, were often found. Caseous matter was therefore asserted to be a devitalized retrograde state of preëxisting gray tubercle. Tubercle corpuscle was designated the product of "specific inflammation," an exudation corpuscle modified by tubercular taint, unfitted for organization and undergoing caseation. But Virchow has established the fact that caseation is essentially the result of the too close crowding of cellular elements, a natural and frequent result of inflammatory infiltration. Hence a large proportion of all cases of pulmonary phthisis are of non-tubercular origin, the sequelæ of inflammatory thoracic diseases.

The gray tubercle was still considered a neoplasm, the product of tubercular dyscrasia, a structure predetermined by the specific vice of the blood.

[1] "Ann. d. Berl. Char.," 1850, p. 362.

[2] "Traité des Mal. Scroph. et Tuberc." ("Lehrb. d. Scrophel- und Tuberkelkrankh.," 1851).

[3] Early edition of Walshe on "Diseases of Lungs."

Piorry said, in 1860, "Pulmonary phthisis is a combination of multifarious, variable phenomena, and not a morbid *unity*."[1]

Niemeyer,[2] though not an originator of the more recent views of the pathology of phthisis, as a systematic author on medicine, has been largely instrumental in establishing their recognition. To the American and English physician who considered "tuberculization" a first stage of phthisis, the proposition that phthisis most often existed without tuberculosis, but that tuberculosis was liable to supervene in established phthisis, seemed false and confusing. The confusion was one only of names. Niemeyer meant by phthisis the presence of inflammatory consolidations in the lungs, and that these, when caseated, were liable to develop in adjacent or distant parts a vitiated new formation—gray tubercle. What is gray tubercle? As late as 1871 it was said by Walshe,[3] "Attempt to fix the microscopic nature of tubercle is impossible." On the contrary, in 1873 we are told[4] "the knowledge of tubercle is now tolerably complete, and that there are no elements in the induration of phthisis not to be found in the healthy lung."[5]

Robin, Empis, Rokitansky, Rindfleisch, and others, regard gray tubercle as a hypergenesis or inflammatory overgrowth of normal pulmonary structures, especially the connective tissue, or as granular aggregations of exudation substance. Bouchut[6] describes tubercles as "fibro-plastic granulations;" and Empis[7] proposes for miliary tubercle the name "granulie," and for tuberculosis the "granular disease."

[1] Paper before the French Academy, about 1860.

[2] Niemeyer, American edition, "Text-book of Practical Medicine," vol. i., chap. xiii.

[3] Walshe, seventh London edition, 1871.

[4] T. Henry Greene, "Introduction to Pathology and Morbid Anatomy," London, 1873.

[5] Burdon-Sanderson, Reports of Pathological Society of London, *Lancet* of August, 1873.

[6] "Traité des Maladies des Nouveau-nés," etc., Paris, 1862.

[7] "De la Granulie, ou Maladie Granuleuse," Paris, 1865.

Experiments for the development of tubercle by artificial infection have led to the assertion of a still more definite structure. Villemin, in 1865, Simon, Fox, Sanderson, Cohnheim, Klebs, and many others,[1] in 1867 and 1868, successfully developed tubercle in animals by the introduction to the circulation of various foreign septic matters, as well as soluble inflammatory products. The deduction from these experiments was, that purulent or caseous matter in the lungs or in any part of the body might, if absorbed, infect or contaminate the blood, and develop tuberculosis, the same as when inoculated. Infection explains why phthisis is frequently developed by the blood-disorganizing fevers, by the protracted suppuration of wounds and abscesses, by caries and necrosis.

The process of infection is considered to have a confirmation in the advanced knowledge of the histology of the lymphatic system.

Baillie,[2] in 1794, mentioned the resemblance of gray tubercle to gland-tissue. Williams,[3] in 1828, believed its uniform shape and size were due to some peculiar anatomical structure. Virchow[4] so far noticed the resemblance that he ranked gray tubercle, a new formation, among the "lymphomata." Hérard and Cornil[5] consider it a gland-like neoplasm. The existence of capillary lymphoid tissue in the healthy lungs is an established fact of histology. It is variously designated adenoid tissue, the lymphoid capillaries, the perivascular lymph-canals, and is fully described by Recklinghausen in Stricker's "Histology," of which there is an American translation.[6] That gray tubercle is, in many cases, an *inflammatory development upon this lymphoid tissue*, was first asserted by Buhl[7] as early as 1857. His views are sus-

[1] All enumerated. Wagner's "Allgemeinen Pathologie," pp. 478–485.

[2] *Op. cit*, pp. 36, 37.

[3] Stated by Dr. Williams, *Lancet*, August and September, 1873.

[4] "Cellular Pathology," 1857.

[5] Hérard and Cornil, "La Phthisie Pulmonaire," 1867.

[6] "Manual of Histology," by Prof. Stricker, p. 215, etc.

[7] "Zeitschrift f. Rat. Med.," 1857, vol. iv., p. 49.

tained by Billroth, Frey, Klebs, Fox, Sanderson, and others, who consider miliary tubercle a reticulum or net-work of lymphoid tissue, whose apparent nuclei are, in fact, exudation or white blood-corpuscles, or nuclei developed by their proliferation. Histology recognizes also the termination of the perivascular lymphoid canals in certain lymphoid spaces of the walls of the vesicular alveoli. Schüppel[1] believes tubercle may be an inflammatory infiltration of these spaces, developing "giant myeloid cells"—large, multinuclear masses of protoplasm, of irregular contour, and having processes directly connecting with the lymphoid reticulum of the blood-vessels. Two facts, unexplained by any other theory of tubercle, would seem elucidated by the office of the adenoid tissue: 1. That miliary tuberculosis, without caseous masses, is so frequent in infants and young children; 2. That miliary tuberculosis, whether in children or in adults, so often leads to death, without softening or any destruction of lung-tissue. The glandular system of children is susceptible to very slight irritations and to impoverished states of the blood. The preservation of form and consistency is the result of definite structure.

This view of the pathology of tubercle does not ignore the greater liability to phthisis of those who have an unfavorable family record, an inherited defect of constitution. But it denies an exclusive or specific dyscrasia, and explains many cases of tuberculosis, in those who had robust ancestors and previous personal health and vigor, from extreme violations of hygiene, from depressing disease, surgical causes, etc.

These views are held not only by their German originators (too often rewarded for their scientific research by the stigma of "extremists"), but by leading authorities in England, by Fox, Sanderson, Powell, Greene, Williams, Pollock, and Bastian.

Etiology.—We recognize *predisposing* and *exciting causes* of phthisis.

Many have regarded specific *tubercular diathesis*, or *hereditary taint*, as the only *predisposing* cause. Others have

[1] "Lymphdrüsen-Tuberculose," Dr. Oscar Schüppel, Tubingen, 1871.

taken a middle ground; they recognize the tubercular diathesis in many cases but claim a relationship of struma or scrofula, as a predisposing cause of many more. Modern etiology asserts that scrofula and tuberculosis are one in nature, if not in degree, and denies a peculiarly tubercular diathesis. Even Louis recognized the lymphatic temperament as predisposing to phthisis, but in no direct and true sense, for struma, scrofula, and impoverished states of the blood, were considered impotent to produce phthisis, unless a true tubercular diathesis preëxisted. Walshe[1] condemns the idea that "a 'below par' state of health favors the essential vice of nutrition developing tubercle." The old idea is thus expressed by a modern advocate:[2] "It may be enunciated as a fact that every child of a consumptive parent is, from its birth to its death, threatened with the same complaint."

When a disease is unusually prevalent, it is natural to suspect communicability, rather than varied and common causes. Thus phthisis was once regarded as contagious. Morgagni, in 1760, feared to study its lesions; Portal would not attend autopsies of the tubercular. Others considered it contagious only among relations or persons closely associated; and at the present day, in some communities, the bedding of the deceased consumptive is burned. A far better explanation of the prevalence of phthisis was found in hereditary taint.

But, the recognition of the inflammatory forms of phthisis at once set apart a large class of cases as of non-specific origin. Aitken says, "In each generation much phthisis is non-hereditary." And, finally, of the truly *specific nature of any class* doubt exists, strengthened by the writings of Simon[3] and others on the relationship of scrofula. It is a significant fact that the nomenclature of diseases adopted by the Royal College of Surgeons[4] includes in a common group, termed

[1] "Diseases of Respiratory Organs," fourth London edition, p. 464.

[2] Inman, "Restoration of Health," London, 1870.

[3] "General Pathology," first edition, Lecture ix.

[4] "Nomenclature of Diseases," constitutional class.

the "tubercular order," scrofula and phthisis. The two dyscrasiæ are thus ranked as members of one family.

All efforts to prove, by statistics, the existence, in a majority of the consumptives, of an unfavorable tubercular family record, have failed.

Dr. Cotton[1] analyzed 1,000 cases at the Brompton Hospital, and could prove hereditary taint in but 367. Scott Allison's observations, at the same institution, were equally negative.[2] Walshe, by careful inquiry among the phthisical, concludes that not over 26 per cent. have had parents affected with phthisis. M. Pidoux[3] says "not over 25 per cent. of those born of consumptive parents themselves become so."

But let us remember that the occurrence of many cases of pulmonary phthisis, in the successive generations of a family, is not a proof of a specific cause. Possibly all were of the simple inflammatory form, with no connecting link. It is easy to understand why phthisis is so universal; it is liable to result whenever the health is depressed, the nutrition is impaired, or the blood is invaded by septic matter. Waller,[4] Cohnheim,[5] and others, have shown that all structures are developments of preëxisting elements of the blood. All new cells, whether they grow to normal tissues, or terminate in morphological products, are at first leucocytes. There is then no "tubercle corpuscle," no "typhoid cell," no "pneumonic or exudation corpuscle"—each is the same white blood-corpuscle whose subsequent changes are the result of healthy growth or pathological aberration.

So, of tuberculosis, Simon[6] says, "The child inherits an imperfect pattern of development, ... a disposition to form blood in a manner which shall give tubercle as a collat-

[1] Richard Payne Cotton, 1858.

[2] Paper in 1848, "Observations at Brompton," quoted by Aitken.

[3] Quoted by Dr. Durant, Paper on "Curability of Phthisis," New York Medical Society Transactions, 1871.

[4] *Philosophical Magazine*, 1846.

[5] Virchow's "Archives," vol. xl., 1867, p. 1.

[6] "General Pathology," American edition, 1852, p. 128.

eral phenomenon." He regards the scrofulous deposit in the cervical and other lymphatic glands as identical with the material of pulmonary and other visceral tubercle; scrofula, though far more prevalent, he ranked as of lower degree, and as a soil in which tubercle may grow. The latest pathology and the most recent authorities approve and emphasize this view. In the words of Billroth,[1] "only the tendency to chronic inflammation ending in suppuration and caseation is hereditary; the scrofulous diathesis, not the tubercular, is hereditary." In the words of Paget,[2] "the relationship between the two is that scrofulous constitution implies a peculiar liability to the tuberculous diseases." Again: "We believe," says Wilks,[3] "that tubercle is the secondary form of the disease of which scrofula is the primary form." But the denial of a *specific diathesis* is not the denial of hereditary influences, nor of acquired dyscrasia, as predisposing to phthisis. Thus of the lymphatic temperament and lymphoid origin, Bastian[4] says, "A man may inherit from his ancestors lungs which contain, within themselves, the elements of weakness—organs, the tissues of which are so constituted, that the very slightest developing causes suffice to initiate a set of changes which terminate in one or other of the forms of pulmonary phthisis." Of scrofula says Wilson Fox: "In a family of strumous children, one may have enlarged glands due to carious teeth, another has tubercular meningitis, another later in life has phthisis, following pneumonia or catarrh; the cases and pathological conditions are alike except in result."

It is thus conceded that, in mal-nutrition of the blood, histological elements are impressed with an imperfect type of organization, and result in the low products of struma and tubercle. Therefore, we must look for the *predisposing* causes of pulmonary phthisis among the known existing

[1] Billroth's "Surgical Pathology," American translation, p. 383.

[2] "Surgical Pathology," pp. 715, 716.

[3] "Pathological Anatomy," p. 188, Wilks & Moxon, London, 1875.

[4] "Discussion before the Pathological Society of London," *Lancet*, August and September, 1873.

causes of mal-nutrition—bad air and food, bad assimilation and excretion, and all violations of sanitary law.

The lower animals, when unnaturally confined, develop phthisis. M. Hazard[1] found that cows in the stables of Paris often were consumptives. Dr. Crisp[2] states that phthisis was once frequent among the cows of London, but has been greatly lessened by sanitary changes. Simon found the animals in the Zoölogical Gardens frequently dying of phthisis, including many species free from this disease in their natural state.

Dr. Crisp, in repeated examinations, found no tubercle in the lungs of animals dying in their habitat.

Dr. Henry MacCormac,[3] of Belfast, is the well-known advocate that "rebreathed air" is the chief cause of phthisis. Dr. Pollock[4] considers "deficient ventilation and crowded apartments eminently productive of tubercular disease." Dr. Christison's[5] statistics show the depressing influence of city life, especially among the lower classes. In Edinburgh, as compared with the country, the mortality from all causes was as four to three, the deaths from phthisis were nearly as two to one. The annual mortality from phthisis in a population of 100,000 was, in Glasgow, 385; in Edinburgh, 283; in the Highlands, 179; in the Lowlands—agricultural districts—104 to 138. Waters,[6] of Liverpool, and many others, give the same testimony. Vitiated air is not the only cause of the greater prevalence of phthisis in cities.

The relative inactivity of city-life, and the constraint of many of its pursuits and trades, result in deficient chest-expansion and diminished volume of the lungs. The protective influence of active exercise is marked. M. Lombard found phthisis occurring in 141 of those whose life was sedentary;

[1] "Annales d'Hygiène Publique."

[2] Pathological Society, London, 1873; *Lancet*, September.

[3] "Consumption as engendered by Rebreathed Air," second edition, London, 1865.

[4] "Prognosis in Consumption," London, 1865.

[5] "Address before the Social Science Association," Edinburgh, 1863.

[6] "Diseases of Respiratory Organs."

in 89 of those more actively engaged. The chest-expansion may be regarded a determining cause in resisting and keeping inoperative many combined unhygienic influences which, with neglect, would lead to pulmonary disease. Phthisis is well known to attack those parts of the lung which in health, for anatomical and physiological reasons, expand least. When the constitution is enfeebled or subjected to deleterious surroundings, such parts are least able to repel disease, by reason of slow circulation and often passive congestion. The apex of the lung is the part which is least expanded, and most often the seat of consolidation. In 4,530 cases examined by Pollock, deposit began at the apex in all but 64.

Certain trades cramp the chest; others poison the atmosphere and develop dyscrasia; many more, by volatile emanations and irritating particles, produce catarrhal and interstitial inflammations.

M. Lombard, Marc d'Espré, and Benoiston de Château-neuf, have variously stated the mortality from phthisis in the poor as twice or three times that among the rich. Undoubtedly the knowledge and observation of hygienic principles among the rich add to their health and longevity. The poor suffer not only from their poverty, but also from their improvidence, their ignorance, and neglect of the simplest sanitary laws. That extreme privation and constant errors in quantity and quality of food conduce to develop or intensify dyscrasia, will hardly be questioned. Bennett[1] and Dobell[2] go further—the former assigning acidity of the alimentary tract, the latter deficient pancreatic secretion, as a starting-point of phthisis. They agree in the belief that failure of the intestinal fluids to digest fats causes a deficit in the chyle, incomplete elements of the blood, and results in tissues capable only of retrogression, since they are wanting in the normal proportion of fatty to albuminoid substance. Their views, however, are not established.

We have reviewed the chief predisposing causes of phthisis.

[1] "Pulmonary Phthisis," 1853.

[2] "Tuberculosis," second edition, London, 1866.

It remains to consider the *exciting causes*—the inflammatory thoracic diseases.

Louis affirmed that pneumonia, pleurisy, and bronchitis, had no influence in developing tubercular phthisis.[1] But few persons will take so extreme a ground. Watson, in quoting Louis's assertion, as above, terms it a most dangerous doctrine, and says, "Dormant predisposition is often awakened into actual disease, and latent tubercles are often accelerated by inflammation of pulmonary tissue."

As regards pneumonia, it is the general experience that uncomplicated cases, in healthy persons, completely resolve and leave no disease—a remarkable fact, when we remember the frequency of pneumonia, and that a hepatized lung contains about two pounds of inflammatory exudation.[2] But it is equally true that in periods of influenza, when broncho-pneumonia develops, and in seasons when pulmonary inflammation assumes a typhoid type, we have not only a greater fatality, but sequelæ which often advance to phthisis. When pneumonia is interstitial, and its resolution is incomplete, we have material for pathological action.

The special influence of bronchitis in the production of phthisis has been and is denied by many. Louis said, "The female sex, most disposed to phthisis, suffers least from bronchitis."

Waters thinks cold and damp are minor causes, since phthisis preferably attacks the in-door worker.

The tables of Buchanan, of Forry, and others, are intended to show that phthisis is most fatal inland, and less prevalent on coasts, where bronchitis is most common. Such statements and statistics do not disprove the causative influence of bronchitis. Thoracic inflammation, in any form, is harmless, if the vitality of the system insures a speedy and perfect resolution; danger arises only from permanent infiltration of the tissues, or accumulation of exuded elements in the smaller tubes and the vesicles. Bronchitis occurs at one or many

[1] Watson, p. 723, American edition.

[2] Investigations of Dr. Stiles's paper, *New York Medical Record*.

periods of nearly every person's life. It is the power to surmount it and avoid its frequent recurrence, which determines its insignificance or its gravity. The male has better health, a more robust physique; the out-door worker has active circulation and expanding lungs; the dweller on coasts breathes a more stimulating air. In such subjects, bronchitis more certainly ends in recovery, speedy and complete, less often lapsing into subacute and chronic forms.

But argument is unnecessary, since pathological demonstrations are frequent of broncho-pneumonia, the impaction of catarrhal products, the occlusion of smaller bronchi and degeneration of lobules, and general infiltration of the fibrous framework of the lungs—following the bronchitis of the young, the old, the feeble, and the subjects of asthenic disease and slow convalescence.

Irritation of the throat, by continued cough, causes bronchial hyperæmia and bronchial catarrh. Hence throat-diseases may contribute toward the ultimate condition of phthisis.

Next to the lungs, tubercle, Billroth tells us, is most frequently located in the larynx.

The *post-mortem* frequency of pleuritic adhesions has always been noted. In Matthew Baillie's "Morbid Anatomy," 1794, they are termed "the most common morbid appearance in dead bodies." This is the personal experience of every physician.

Occlusion of a pleural cavity is exceptional, partial occlusion is not infrequent; adhesion bands and fibres, intact or ruptured and atrophied, plastic patches and thickened pleura, are common.

When not absorbed or broken down by the respiratory movements, membranes or bands often organize; they may become vascular; and arteries of size, as traced by Van der Kolk and Guillot, may pass through the connecting adhesions, from the lung to the thoracic wall. The clinical frequency of local and slight pleurisy is attested by Walshe; "evanescent pleurisy," he says, "is of the most frequent occurrence."

Dr. J. R. Leaming, of this city, in his several published monographs, has put on record many cases of local dry pleurisy, and detailed the physical signs by which they are to be positively recognized.

Laennec, in 1819, asks, "Can tuberculosis be a termination of pleurisy?" and adds, "The proposition is absurd, for it is preposterous to suppose that inflammation of one organ should terminate in another."

Yet many clinical records testify to the occurrence of local pleuritic pain, and confirmatory physical signs of local pleurisy, preceding any symptoms or physical signs of pulmonary deposit, and when chest-expansion and perfect vesicular element of respiratory sound proved the lung to be intact.

Conceding, as is generally held, how often pulmonary tubercle is primary and leads to a local and secondary pleurisy, does not the frequency of local and evanescent pleurisy, and the common occurrence of adhesions in the cadaver, justify the opinion that in many cases they may be the antecedents and cause of phthisis? Louis[1] found, in 112 cases, only one in which the lungs were free throughout their whole extent. Broussais[2] claimed to have seen pleurisy the precursor of tubercle in many cases.

Swett[3] thinks that phthisis is more often due to pleurisy than to pneumonia or bronchitis.

Dr. Leaming[4] is well and widely known as an advocate of the frequent pleural origin of phthisis.

Rindfleisch,[5] under the head of "Pyrogenous Pneumonia," concedes that "pleurisy may precede and be the cause of pulmonary infiltrations;" the outer row of infundibuli are often filled with exudation.

Histology and pathology, as well as observation of symp-

[1] *Op. cit.*

[2] "Chronic Phlegmasiæ," vol. i.

[3] "Diseases of the Chest," 1856.

[4] "Plastic Exudation within the Pleura."—Dr. Brown-Séquard's "Archives," March, 1873.

[5] "Pathological Histology," p. 417, American edition.

toms and physical signs, will support this view. There is an intimate vascular connection of pleura and lung, and through established adhesions often there is active collateral circulation; thickening and adhesions of the pulmonary pleura result in irritation and hyperæmia of the lung. Persistent hyperæmia results in hyperplasia or in infiltration, which may degenerate and become crude tubercle. The influence of pleural adhesions, by causing immobility of the lung, in favoring tubercular processes, is stated in the recent revised edition of Jones and Sieveking's,[1] though regarded essentially secondary.

The usual location of phthisis, at the apex, accords with the view that pleural adhesion may sometimes be its cause. By inflammation, the pleura is engorged and tumefied, is deprived of its epithelium, and presents a villous, granular surface, whose opposed walls are liable to unite.

This may occur at once when exudation is limited to subserous infiltration. But, as a rule, there is an escape of serum or sero-plastic matter into the pleural cavity. The liquid exudation gravitates and keeps the middle and lower surfaces separated, but the upper are collapsed, in apposition, and unite their granular, villous processes. This process of pleuritic adhesion Rindfleisch compares to the union of the opposed walls of a granulating wound. Such adhesions, when formed, may be speedily ruptured and absorbed; but, when permanently located and organized, they are a source of irritation and restraint to the lung through the person's future life. Will not an irritation of lung-surface, renewed eighteen to twenty times per minute, result in pulmonary hyperæmia and progressive consolidation? Will not the extent of peripheral nerve-irritation in pleurisy tend to depress the general health just as superficial burns and scalds are a source of shock, and extensive irritable ulcers lower the innervation of the whole body?

The reduction of the lung to a cirrhotic, carnefied state

[1] "Pathological Anatomy," p. 511, Jones and Sievking, by Payne, London, 1875.

by contracting thickened pleura, the presence of caseous nodules in masses of pleuritic exudation, are less frequent but recognized dangers of pleurisy.

As inflammation of the cerebral meninges, the pericardium and peritonæum are sources of injury to the organs they invest, so the pleura when inflamed may result in consolidation of the lung.

Climate, whose extremes and fluctuations are the immediate causes of these thoracic inflammations, has a direct relation to the prevalence of phthisis. It is a disease of temperate regions, but little known in the colder and more northern ones, and comparatively infrequent in tropics. It prevails where the temperature is most changeable. Removal from warm to colder climates, as from the Indies to Europe, or from the Southern to the Northern States of the Union, often develops it.

The United States census for 1870 shows the geographical correspondence of the distribution of phthisis with the existence of cold and changeable climate. In the different parts of our country, the mortality from phthisis varies between one-fortieth and one-fifth of the deaths from all causes—the greater mortality coinciding with the colder localities and those most subjected to climatic change. This report affords a controversion of the statistics of Forry and others, as to bronchitis. We find in California a seaboard district having a maximum mortality from phthisis, and an adjacent interior where the disease attains a minimum.

"Phthisis ab hæmoptoë" was an expression frequently and fallaciously employed, previous to the scientific physical exploration of the chest; and yet the term is applicable to many cases of consumption, in which ill-health and pulmonary deposit, as indicated by physical signs, were preceded and induced by an attack of hæmoptysis. Bronchial arteries, as demonstrated by Cammann,[1] by Waters,[2] by Schultze,[3] and

[1] *New York Journal of Medicine.*

[2] Prize essay on "Anatomy of the Lungs."

[3] Article on "The Lungs," Stricker's "Histology."

others, return only a part of their contained blood to the bronchial veins; the remainder passes—not by capillary anastomosis—but by direct intercellular arteries to the branches of the pulmonary vein, and to the left side of the heart.

Hæmoptysis—usually bronchial blood—may occur in perfectly healthy persons, whose lungs are intact, from excessive effort and extreme cardiac excitement, but more often, the lungs being healthy, as a result of mitral insufficiency. Again, any pulmonary deposit, though of a harmless nature in itself, may obstruct these intercommunicating vessels in adjacent tissues, causing a local engorgement of a bronchial twig, and hæmoptysis. Hæmoptysis, from conditions of local congestion, is often a relief or positive benefit, provided clotted blood be not retained in the bronchi.[1] The lodgement of blood in the bronchi, from any cause, is disastrous to the integrity of the lung, exciting local broncho-pneumonia, and exerting a septic influence on the general health; rapid infiltration and caseation are its frequent and fatal sequelæ.

The danger is well illustrated by the fact that bronchopneumonia often results from the accidental entrance of blood into the bronchi, when tracheotomy is accompanied by hæmorrhage, or any operation upon the throat is performed under anæsthesia. Profuse hæmoptysis demands arrest, but a slight attack is less to be feared than the dangers incident to hastily checking it—the development of a new focus of caseous tubercle.

Having thus reviewed the pathology and etiology of pulmonary phthisis, as a basis of prevention and early arrest, we may be guided by two conclusions:

1. Dyscrasia, or predisposition, is largely the cumulative result of depressing influences, which sanitary control and personal regimen may diminish.

2. Inflammatory attacks are the chief exciting causes of pulmonary phthisis, whether in the strumous or in the previously healthy; causes which may be largely averted by selection of climate, and avoiding the exposures which lead to

[1] Niemeyer, vol. i., chap. xiii.; Leaming, "Hæmoptysis."

them, or rendered trivial by constant care and supporting treatment.

Prevention.—The prevention of phthisis is a question—

1. Of public hygiene.

2. Professional supervision of persons and families in whose cases predisposition—inherited or acquired—exists.

1. All public sanitary measures, as the prevention of overcrowding, the vacation of unhealthy abodes, the enforcement of tenement-ventilation, the correction of defective sewerage and drainage, the public inspection of food, and the removal of all known causes of blood-poisoning, will have an ultimate influence in lessening the extent of the dyscrasia, in correcting the lymphatic temperament, struma, or scrofula, and an immediate influence in preventing the asthenic diseases and low types of thoracic inflammations, which may develop, during convalescence, the degenerative processes of phthisis. The sanitary reforms in London have increased the average longevity from thirty-five to forty-one years.[1] The favorable effects of hygienic conditions upon the very worst class of lives may be seen by a study of the various public institutions for poor children. Asylums for foundlings, for orphans and half-orphans, contain the offspring of parents who have died of phthisis and of other diseases engendered by privation and vice: reclaimed from homes of poverty and neglect, they have inherited all the conditions of struma. Yet in one institution there will be a constant recurrence of scrofulous disorders and frequent deaths from miliary tubercle of the brain, the lungs, or bowels; while another asylum may present a striking contrast of general good health, and an exceptional occurrence of tuberculosis.

In a miniature community, then—through the agency of diet, clothing, exercise, and proper housing — meningitis, phthisis, scrofulous glands, eczema, and purulent ophthalmia, have been measurably controlled.

In the community at large, public hygiene will likewise lower the scrofulous or tubercular diseases, exerting a marked

[1] Since 1600. Dr. Stephen Smith, "Public Health Reports," 1875.

effect on the susceptible organism of growing children, and a corresponding influence upon adults. Of our adult foreign population, how many there are who can date their pulmonary trouble from the noxious exposures of the ship, and landing at a season and in a climate whose severities of temperature they were not prepared to withstand! Native and foreign alike suffer from the defects of the tenement-house, the factory, and the shop. The places where the poor dwell and work must be purified by light and air. There are questions of public hygiene equally vital to the rich; such are the proper location and building of the home, the town, the city, proper sewerage, the purity of the water-supply, and the hygiene of the public schools.

2. The guidance of the individual for the prevention of phthisis may begin at birth—the nursing and airing of the infant, the diet of the bottle-fed and weaned, the management of first dentition, the treatment of summer diarrhœa, and the eruptive fevers, so as to leave no catarrhal sequelæ or reduction of vigor.

But, in those in any way predisposed, it is at adolescence, in approaching maturity, that advice and direction should avail most—to the too-assiduous student, the youth ambitious for business advancement, the young woman entering society. The family history may well be reviewed, the personal temperament and physique considered, the safety of the proposed vocation estimated. I believe it a duty to dissipate the discouragement which often exists through a belief in the power of a specific, hereditary tubercular taint. But it is no less a duty to clearly depict the dangers which arise from incorrect habits of living, neglected action of the lungs, and colds; and to cast a horoscope of the future which is in store, unless the preservation of health is a constant study. I can recall many instances of most decisive results from such advice. Directions as to personal regimen have been followed in detail. The diet is to be judiciously selected, easy of assimilation, and in quantity avoiding the excess which taxes the digestion and loads the alimentary canal, since chronic dyspepsia and con

stipation impoverish the blood. The value of a healthful state of the skin cannot be over-estimated—not alone its cleanliness and functional activity in elimination, but rather its normal temperature and perfect circulation, as protections from visceral congestion. The statistics of Edward Smith[1] on the protective influence of proper clothing accord with my experience in several recent cases, in which covering the general surface in flannel accomplished a tonic effect which other means had failed to obtain. The general clothing should be suited to the season, with provision for adapting it to variations of temperature from day to day.

No physician, who has not made it a business to inquire closely, can be aware of the irregularities, the errors, and neglect of the proper protection of the body which exist in every class of society. Injudicious and too frequent bathing is dangerously sedative to those who are strumous or in delicate health, and lack the vitality for vigorous reaction. The baths of such persons should be stimulating, by the addition of sea-salt, and followed by thorough friction.

The full development of the chest, and the increase of its expansive measurement to a proper standard, must be accomplished either by systematized methods of chest-exercise and inhalation, or by a business pursuit which secures the same result. The flat surface must become rotund, and feeble respiratory sound be replaced by the rhythm and volume and the vesicular element which characterize its healthy state.

Exercise and out-door life must be secured in choosing a vocation—the life of a farmer or horticulturist, the profession of mining or civil-engineer. The gymnasium is less valuable than active work, walking, athletic sports, and riding on horseback. Sydenham is reported to have said, "In the treatment of consumption the best physician is a horse, and the best apothecary is an ass."[2]

It is too often true that, in advanced stages of pulmonary phthisis, the physician can do little to control its progressive

[1] Edward Smith, "Consumption, its Early and Remediable Stages."

[2] Motherby's Dictionary of Medicine, "Consumption."

tendencies. But its early arrest, when presenting its first distinctive symptoms and physical signs, is to be attempted, and in very many cases, through persistent effort, will be accomplished. There are both methods of procedure and medicines which cure, or control the progress of, the incipient stages of phthisis. These stages are varied in nature—in one instance being a purely local lesion of the lung, in others complicated by dyscrasia. Manifestly, the treatment in either state must never be other than supporting. To increase the nutrition of the blood, lessens the danger of new inflammatory deposits, and the liability of those already present to degenerate. The arrest of phthisis, when possible, will be chiefly by three agencies:

1. *Food*—including those articles which are most highly nutritive, and those remedies which conduce to its thorough assimilation.

2. *Chest-expansion* as a means of fortifying the lung against renewed inflammatory attacks.

3. *Climate.*

The diet must possess the qualities already enumerated in considering prevention, and digestion will require no less the stimulus of air and exercise. These may suffice. Autopsies of aged persons show in many cases the proofs of spontaneous cures. But cod-liver oil, as an agent of nutrition, is recognized as most curative of all remedies for phthisis. It is the richest of the hydro-carbons. In dynamic, or force-producing power, as estimated by Pavy,[1] it takes precedence of all other aliments. Cream will not nourish so rapidly. Pancreatic emulsion is inferior in benefit. It has no substitute. Of its mode of action there are many theories. Certainly it increases the general nutrition of the blood, for its use extends to other diseases than phthisis.

The residents of northern coasts who subsist largely on fish and fats are reputed free from tuberculosis. Bennett claims a perceptible decrease in the mortality from phthisis in Scotland since the use of oil in its treatment.

[1] Pavy on "Food and Dietetics," London, 1874.

Cod-liver oil may often have its efficacy increased by other remedies. Phosphates may be incorporated with it, as supplying an element of normal nutrition. Pancreatine will insure its digestion and absorption. Alcohol will aid its assimilation in greater quantity.

Campbell,[1] Thompson,[2] and Walshe, state that ozone or oxygen, incorporated with oil, lowers the frequent pulse fifteen beats, and aids nutrition. Iodine, a well-known alterative in scrofula, may be employed when diathesis exists. Tonics of every kind coöperate in the nutritive treatment.

Chest-expansion is the great means of securing the functional activity of the uninvaded portions of the lung, and insuring the oxygenation of the blood.

Climatic treatment has for its chief purpose a studied avoidance of colds, and their speedy resolution when acquired.

Changes of residence, either temporary or permanent, with reference to the benefits of climate, are permitted by the circumstances of but few consumptives. We are forced, therefore, in the majority of cases, to combat at home the inflammatory disorders which our severe winters excite. Ammonia and quinine are preëminent remedies in accomplishing this purpose. Ammonia is a diffusive stimulant, is almost exclusively eliminated by the lungs, and favors the escape of hyperæmic products in the form of mucus.[3]

Quinine limits exudation, reduces temperature and danger of caseation, and hastens absorption of inflammatory products.

[1] Walshe, "Diseases of the Lungs."

[2] "Braithwaite," part xi., p. 294.

[3] Woods's "Therapeutics," Philadelphia, 1875, *et al.*